BioNanoSci. (2017) 7:633–635
DOI 10.1007/s12668-017-0435-x

Autoantibodies Against dsDNA Modulate Contraction of Blood Clots

Alina D. Peshkova[1] · **Giang Le Minh**[1] · **Izabella A. Andrianova**[1] · **Rustem I. Litvinov**[1]

Published online: 31 July 2017

Abstract The degree and rate of clot contraction (retraction) in systemic lupus erythematosus (SLE) patients, especially in those with a high level of anti-double stranded DNA (dsDNA) antibodies in the blood, was significantly reduced compared to healthy donors. We hypothesized that this effect was caused by the anti-dsDNA antibodies. To test this assumption, we investigated the kinetics of blood clot contraction in vitro in the absence and presence of anti-dsDNA antibodies purified from the blood serum of SLE patients. The degree of clot contraction was increased immediately after addition of the anti-DNA antibodies in a concentration-dependent manner. This stimulating effect was abrogated by a monoclonal antibody against the platelet Fc-receptor. On the contrary, after prolonged incubation (for hours) of the blood samples with the anti-DNA antibodies, the extent of clot contraction was significantly reduced. These results suggest that anti-dsDNA antibodies in SLE induce Fc-receptor-mediated chronic platelet hyperactivation, resulting in platelet exhaustion and dysfunction, including reduced contractility. The impaired contraction of blood clots and thrombi caused by autoantibodies may be an important pathogenic mechanism that affects the course and outcomes of thrombotic complications in SLE.

Keywords Blood clotting · Clot contraction · Systemic lupus erythematosus · Anti-DNA antibodies

✉ Alina D. Peshkova
alinapeshkova@list.ru

[1] Kazan Federal University, 18 Kremlyovskaya St., Kazan 420008, Russia

1 Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that affects multiple organs and tissues, resulting in diverse symptoms and outcomes. It is characterized by production of antibodies (Abs) against a variety of autoantigens, specifically against double stranded DNA (dsDNA) [1]. Platelets are a well-known target for anti-phospholipid Abs [2] that alter platelet function in SLE [3] and predispose to thrombosis [4]. It is unclear whether anti-dsDNA Abs can also contribute to thrombotic complications in SLE. Blood clots and thrombi are known to undergo volume shrinkage driven by contracting platelets, the process named clot contraction or retraction [5, 6]. Despite its potential clinical importance in modulating vessel obstruction and blood flow, the mechanical remodeling of blood clot and thrombi has been underestimated and understudied [7]. Here we studied the effect of anti-dsDNA Abs isolated form the blood of SLE patients on the ability of platelets to squeeze blood clots.

2 Materials and Methods

Blood from SLE patients and healthy donors was withdrawn using a protocol approved by the Ethical Committee of Kazan State Medical University. Only patients who were not treated with anti-coagulants or anti-platelet drugs were included in the study. Blood samples of 37 SLE patients were analyzed, of which the activity of anti-dsDNA Abs was less than 100 ME/ml in 23 (63%) and > 100 ME/ml in 14 (37%). Blood samples from 60 healthy donors were used as a control. The SLE patients and control subjects were comparable by the age (37 ± 2 vs. 34 ± 2 years, respectively) and sex (women comprised 79 vs. 69%, respectively).